she had an incarcerated hernia, was operated upon and recovered, but developed nephritis and a recurrence of the fibrinous bronchitis. Finally, loss of compensation came on, and the patient died without further fibrinous expectoration. Habel made examinations of the casts in this and another case, also secondary to valvular disease. Stained by Weigert's method, they showed very few fibrin fibres, most of the casts not taking any stain (lithium carmine). Chemical examination also showed the absence of fibrin, but proved that the casts were made up chiefly of mucin. The casts were of acid reaction, and the author thinks this is the cause of the coagulation. According to his view something, probably the action of bacteria, causes the bronchial secretions to become acid. The mucus then coagulates. He applies the same explanation to the casts sometimes expectorated in croupous pneumonia, and was able to confirm his view in a case of the latter disease. —*Centralblatt für inn. Med.*, 1898, No. 1.

The Serum-test for Typhoid Fever at the Montreal Meeting.—The important papers read go to prove that, however great the scientific interest of the serum-test, its results must be used with caution in practice. WIDAL showed, from the extensive statistics of CABOT, that the reaction is present in a very large proportion of cases. He himself only missed it once in 177 cases. He emphasized the difficulty of diagnosticating clinically all cases of typhoid fever. In doubtful cases he had made cultures from the spleen. By this method he was able to demonstrate the absence of reaction in a case of genuine typhoid fever with relapse. The examinations were negative during both febrile periods, the intervening stage and the convalescence. Such cases are so rare as hardly to affect the value of serum diagnosis, but prove that the agglutinative reaction has nothing to do with immunity. They follow naturally after observations showing that in some cases the reaction comes on very late. Widal believes the reaction is often more intense in the beginning of the attack. It diminishes and frequently disappears from the first period of convalescence, and is relatively rare in patients who have been cured for more than a year. W. GILMAN THOMPSON described observations showing that the reaction often became more intense as the disease progressed. WILSON and WESBROOK related an interesting case in which there was reaction on the twelfth, nineteenth, twentieth, and twenty-fifth days; absent on the ninth, thirteenth, and twenty-first days, without corresponding variation in the clinical symptoms. These observers examined a large number of cases in children, and found a reaction in 76 out of 165. The reaction appeared earlier than with older cases. (The clinical features in these cases are not mentioned.) In one case a mother seven months pregnant had clinical symptoms of typhoid, though her blood failed to react on the fourth and sixth days. The child was born on the sixth day, and two days later gave a marked reaction. The mother reacted on the thirteenth and twentieth days. E. BATES BLOCK gave some interesting and valuable details on the technique, and advocated the use of serum, rather than dried blood. Thompson held that the value of the test was about the same as that of the diazo-reaction, " confirmatory in connection with appropriate symptoms, but misleading if positive reliance be placed upon it;" but MUSSER and SWAN seemed to have had a more fortunate experience. The latter observers called attention to the fact that the presence of the reaction does not lessen the importance of making an accurate diagnosis of the patient's condition as a whole, illustrating this by the history of a patient who might have been thought to have a relapse, but proved to have a meningitis following the typhoid attack.—*British Medical Journal*, December 18, 1897.

Hæmatoporphyrinuria, with the Report of a Case.—OGDEN (*Boston Medical and Surgical Journal*, February 24, 1898, p. 169) gives a concise historical review of the subject of hæmatoporphyrinuria, and reports a personal observation.

Hæmatoporphyrin is a rare constituent of the urine, and is a coloring matter derived from the blood. The first observations on the subject were made by Hoppe-Seyler in 1871, when he found that hæmatin heated with sulphuric acid yielded a compound whose acid and alkaline solutions showed spectral bands unlike any substance he had seen before. To this new compound he gave the name "hæmatoporphyrin." Nencki and Sieler determined its exact formula to be $C_{16}H_{18}N_2O_3$, and the former demonstrated that the only chemical difference between hæmatin and hæmatoporphyrin was that the latter was simply hæmatin free from iron.

In 1880 MacMunn described a pigment which he obtained from the urine of a case of acute rheumatism, and which he called "urohematin." In 1883 he reported other cases of acute rheumatism in which this pigment occurred, and from the similarity of its spectrum to that of hæmatoporphyrin he named the pigment "urohæmatoporphyrin."

Hæmatoporphyrin has been found in the urine in pulmonary tuberculosis, in pleurisy with effusion, acute rheumatism, lead-poisoning, and intestinal hemorrhages. The pigment has been found probably most frequently in the urine of patients to whom sulphonal had been administered. In fact, it seems to occur almost constantly when the drug has been given in considerable quantities, but never when only a single dose (one gramme) has been taken. It has also been found in the urine after the administration of trional and tetronal.

Stokvis has found that it occurs after intestinal hemorrhage. In artificial hæmatoporphyrinuria produced in rabbits by the administration of sulphonal, he found that minute hemorrhages into the mucous membrane of the stomach and intestines occurred. From these areas he extracted hæmatoporphyrin, and he believes that in these cases the blood which had entered the digestive tract was changed into hæmatoporphyrinuria, absorbed, and excreted in the urine.

Ogden's case occurred in a woman, aged thirty-eight years, who had suffered from rheumatism for years, and who had a mitral heart lesion. The patient previous to the appearance of the hæmatoporphyrinuria had had diphtheria. During convalescence she began to have vomiting and diarrhœa, and later pains and loss of sensation in the legs and body up to the waist line. Urine and feces were passed involuntarily. On two different occasions trional in fifteen-grain doses was given for insomnia. It was after the administration of this drug that the dark, pigmented urine containing hæmatoporphyrin was voided, and Ogden believes that the drug was responsible for its